Iodine and Hashimoto's Autoimmune Thyroid Disease

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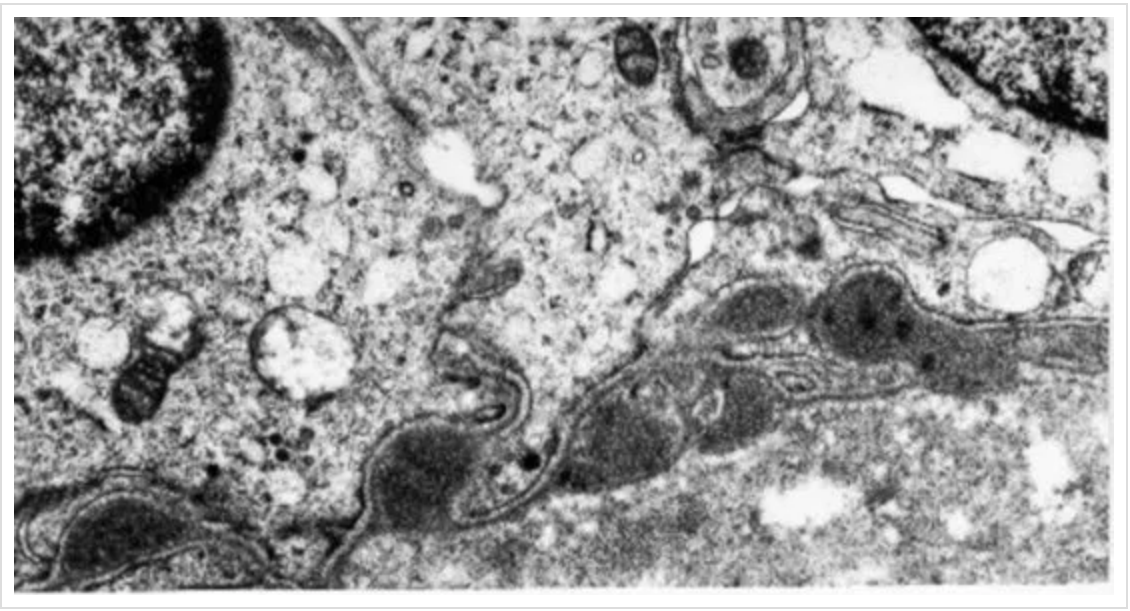


Iodine and Hashimoto's Autoimmune Thyroid Disease
by Jeffrey Dach MD

Above Image:Electron microscopy image of thyroid tissue from a patient with Hashimoto's thyroiditis, showing electron dense deposits of Antibodies (IgG and TG) along the basement membrane of follicular cells. Courtesy of Fig. 2 Endotext Akamizu, Takashi, and Nobuyuki Amino. "Hashimoto's thyroiditis." *Endotext [Internet]* (2017).

Hashimotos Thyroiditis is an autoimmune disease. Microscopic examination of the thyroid gland shows infiltration with lymphocytes and deposition of antibodies on the basement membranes of the thyrocytes (see above image). Clinical examination may show painless enlargement of the gland. Diagnosis is made by laboratory testing showing elevated serum TPO (Thyroid Peroxidase) and Thyroglobulin antibodies. Other antibodies to the NIS, pendrin and TSH receptor may also co-exist.

Thyroid function may be variable. Early stage disease may have normal function. Late stage disease is usually associated with a low thyroid condition (hypothyroidism), as damage to the thyrocytes reduces the ability of the gland to produce thyroid hormone.

In approximately 20% of cases there is spontaneous recovery.(2) How can we predict which patients will recover?

This article is part three. For Part Two, Click Here.

For Part One Click Here

Iodine Restriction for Hashimotos Patients

Dr Yoon from Korea noted that Hashimotos thyroiditis is reversible for some patients.(2) He points out that Hashimoto's was more prevalent in Iodine replete areas that have introduced salt iodination programs (18), and less prevalent in iodine deficient regions, so he speculated that perhaps Iodine levels had something to do with spontaneous recovery from Hashimotos.(7) Dr. Yoon thought Iodine restriction would be beneficial, and he did a study on Iodine Restriction in Korea in 2003 to try to prove his point. (1)

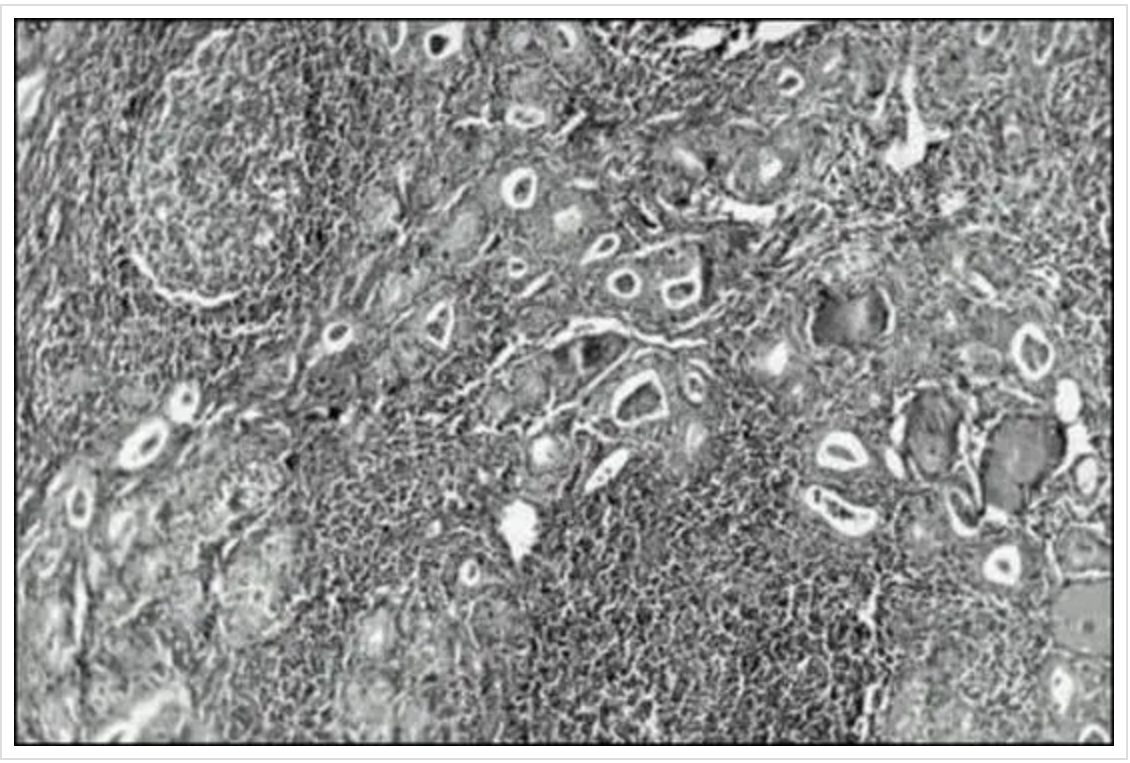


Image 2) Pathology of Hashimoto's thyroiditis. In this typical view of severe Hashimoto's thyroiditis, the normal thyroid follicles are small and greatly reduced in number, There is marked fibrosis. The dominant feature is a profuse lymphocytic infiltrate germinal center formation.

Dr. Yoon's study enrolled 45 Hashimotos patients who had a low thyroid condition (elevated TSH). They were divided into two groups. Group one (23 patients) had iodine restricted diets (less than 100 mcg Iodine /day) . Group 2 (22 patients) had no restrictions on Iodine intake.

Initial labs showed rather high iodine intake for both Group One and Group Two. This is understandable since the Korean diet is high in Iodine.

Group One: 486 mcg. Iodine per day

Group Two: 716 mcg. Iodine /day

Initial TSH for Group one was 37.95, and after iodine restriction (3 mo. later) was 25.66.

Group 2 11.25 and 3 months later (no restriction) was 14.27

According tho Dr Yoon, 78% of Group One recovered a euthyroid state, and 45% of Group Two recovered a euthyroid state. I assume euthyroid means the TSH was in the normal range (0.4-4.0).

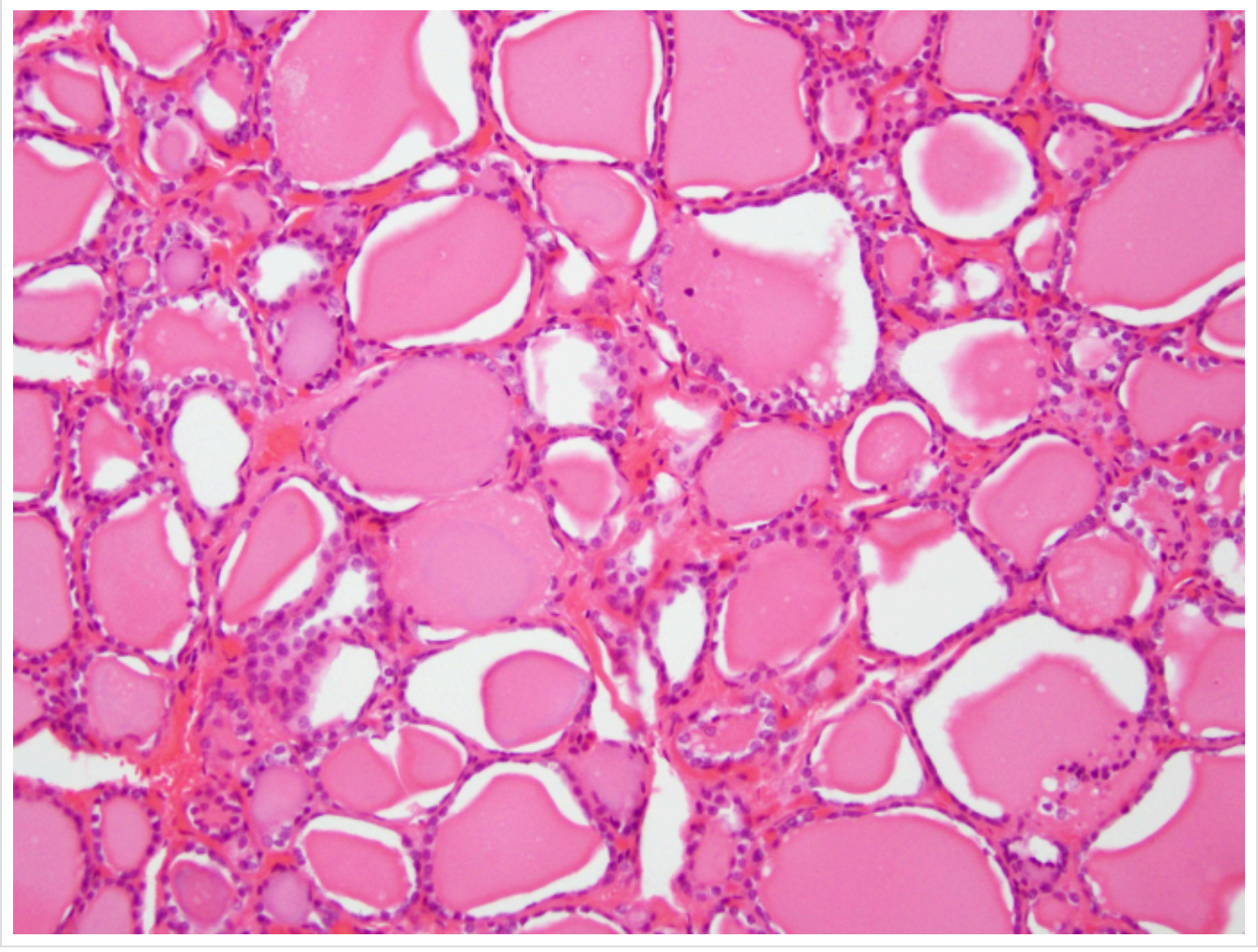


Image 3) Normal Thyroid Histology: Notice the Normal follicles which contain abundant Colliod surrounded by a single layer lining of Follicular Cells.

Iodine Suppresses Thyroid Function, Increases TSH

My first criticism of this study is that Iodine intake, by itself, will suppress thyroid function and cause in elevation of TSH. So in Korea, where dietary Iodine intake is fairly high, some patients may have elevation of TSH caused solely by high Iodine intake, and when Iodine is restricted, the TSH will decline. This effect of temporary TSH elevation from Iodine intake was noted in Peace Corp workers and in Astronauts on the Space Shuttle who used iodine water purification systems.(3-6) In Japan where dietary Iodine ranges from one to twenty milligrams mg per day, elevation of TSH may be caused merely by high dietary intake of Iodine, and TSH values come down to normal when Iodine is restricted.(22)

Iodine Does NOT Augment Autoimmunity

In agreement is Dr. Dayan in his 1996 article on Chronic Autoimmune Thyroiditis in the New England Journal (21):

> *"However, iodine reduces thyroid secretion in both subjects with thyroid autoantibodies and those without thyroid autoantibodies, suggesting that* ***it acts by inhibiting the biosynthesis and release of thyroid hormone rather than by augmenting thyroid autoimmunity.*** *"(21)*

Iodine as a Treatment for Hyperthyroidism

Iodine will suppress thyroid function. Iodine's suppressive effect on thyroid function is so well known that Iodine Therapy with Lugol's Solution or SSKI has been used as a treatment for hyperthyroidism (Graves Disease) for many decades. Surgeons still use it in the hospital as a pre-operative measure prior to thyroidectomy in Grave's Disease patients(10-14)

Back to the Dr. Yoon Study

What Dr. Yoon is calling "recovery from hypothyroidism" is probably just the effect of Iodine on the TSH, which is well known. When Iodine intake is high, the TSH goes up. Removing Iodine in these circumstances causes the TSH to go down.(3-6)

What happens next in the Yoon study is very strange indeed. Dr. Yoon then re-divides the patient group into "Recovery Patients" and "Non-recovery patients", and discusses these two groups. He no longer uses Group One and Group Two. This can be very confusing because the Recovery Patient Group contains both Iodine-restricted as well as NON-Iodine Restricted patients.

Let's look at **Figure Three and Figure Four Data** (Yoon_2003_Figure Three Data) which compares Recovery from Non-Recovery patients.

Table 3. (17 pts.) in Recovery Group (28 pts.) Non-Recovery Group

There were 28 in the Recovery Group in which average TSH of 12 declined to 3, after 3 months of observation. Free T4 increased in the Recovery patients from 0.89 to 1.07 and Free T4 decreased in the Non-Recovery patients from 0.88 to 0.75. The Iodine intake was, on average, **521 mcg per day** (Recovery Patients) and **730 mcg per day** (Non-Recovery Patients).

Look at Table 4. These are the 23 Iodine-Restricted patients

In the 18 pts in the **Recovery Group TSH** declined over three months from 14.2 to 3.18 and Iodine intake was **555.67 mcg/day**.

In the 5 patients in the **Non-Recovery Group,** TSH was high initially 123, and three months later was still high at 106.00. Their 24 hr Iodine intake was **264.92 mcg/day**.

In Table 4, the **higher Iodine intake of 555.67 mcg/ per day** was associated with recovery in 18 patients !!! This is just the opposite of Dr. Yoon's hypothesis that Iodine restriction is beneficial.

My second criticism is: How on Earth can one conclude that Iodine restriction is beneficial when the Recovered patients had **556 mcg/day** of Iodine intake? This is half a milligram of Iodine daily !! This is certainly not Iodine restriction !!

Dr Yoon wanted to predict which patients would recover normal thyroid function and which one would not, so Dr Yoon re-organized the data in Recovered and Non-Recovered patients. As you can see from the above data, there was essentially no difference in Iodine intake when Recovered are compared to Non-Recovered patients. However, Dr Yoon says the higher urinary excretion of iodine in the Recovery patients may explain the discrepancy.

The YOON Study

What is going on in the Yoon study? The Korean diet is relatively high in iodine, and patients with auto-immune Hashimoto's disease are more sensitive to the suppressive effect of dietary iodine, which will increase the TSH level indicating reduction in thyroid function. Restricting the iodine intake in these sensitive people will indeed reverse the suppressive effect of iodine, and TSH will normalize to lower levels. Some may even return to normal range TSH. I have seen this in clinical practice. The sensitivity to iodine in autoimmune thyroid disease is variable, some people are more sensitive than others. In my opinion, the greatest sensitivity can be seen when both Hashimoto's and Graves antibodies are elevated.

What about the Antithyroid Antibodies ??

My third criticism of the Yoon study is that in Hashimoto's patients the parameter we are most interested in seeing improve is the anti-thyroid antibodies. We are most interested in seeing a decline in anti-thyroid antibody levels which indicates improvement in the auto-immune disease.

So, what were the antibody levels initially and after three months in both groups? Was there a significant decline in antibody levels after Iodine restriction. No, there was no difference between the two groups.

How to Make Anti-Thyroid Antibodies Decline

My previous article discussed the numerous studies showing that both Selenium supplementation and, secondly, TSH suppression (with thyroid medication) are the two interventions that consistently reduce Anti-Thyroid antibody levels in the Patient with Autoimmune thyroiditis (also called Hashimotos).

Dr Reinhardt

The effect of iodine supplementation for Hashimoto's patients was studied by Dr. Reinhardt from Germany.

No Effect of Iodine on Antibody Levels in German Study

Dr Reinhardt from an iodine deficient area of Germany examined 40 Hashimotos patients patients given Iodine supplementation (250 mcg/day for four months). Another 43 Hashimotos patients served as controls.(16) The Iodine treated group started with an average TPO Antibody level of 729 and TSH of 7, compared to 483 and 8 for the Control group.

Iodine excretion increased from 72 to 268 in those receiving 250 mcg of Iodine daily. In 7 of the iodine treated patients, further increase in TSH was noted. One patient developed overt hyperthyroidism. Another developed overt hypothyroidism. However, only one patient in the control group had increase in TSH and antibody levels after 4 months.

No Significant Change in Antibody Levels With Iodine Supplementation

In the 40 Iodine treated patients:

> ***"There was no significant change in TPO-Ab levels or thyroid volume during the study period. … Neither TPO-Ab titres nor thyroid volume was of prognostic value in terms of predicting thyroid dysfunction."*****(16)**

One of the 40 Iodine treated patients, a young lady also suffered from Myasthenia Gravis was on Birth Control Pills, and had very elevated antibody levels She developed overt hyperthyroidism which resolved after withdrawal of the Iodine. The authors speculated that this patient may have had underlying Graves disease.(16)

Conclusion:

Dr. Yoon's conclusion that iodine restriction is beneficial for the Hashimoto's patient is not supported by his own data. A reduction in TSH seen in the Iodine restricted group is simply the removal of the suppressive effect of Iodine on thyroid function, with no benefit for the underlying autoimmune process. Dr Yoon's data actually showed higher iodine intake in the Recovery patients. A discrepancy he explains away with the observation this group had higher iodine excretion.

Dr. Reinhard from Germany states that **"there was no significant change in TPO antibody levels in iodine treated patients."** Again this is consistent with iodine having a suppressive effect on thyroid function without any effect on antibody levels.

The Iodine Project

Drs Abraham Brownstein and Flechas

Drs Abraham, Brownstein and Flechas regard iodine deficiency as the leading cause of auto-immune thyroid disease in the US. (25) This is based on the fact that auto-immune thyroid disease has increased to epidemic

proportions during the past forty years, all the while Iodine levels have fallen by 50% based on NHANES government surveys.(23,24) The decline in iodine levels has been related to reduced iodine content in milk and dairy products, the removal of iodine from bread and its replacement with bromine, a goitrogen, the reduced salt intake for blood pressure control, the increased use of noniodized salt etc.(23) Drs Abraham, Brownstein and Flechas have discussed the use of Iodine in Hashimoto's patients and conclude that Iodine supplementation is safe and beneficial in this group in the 6-50 mg per day range.(8,9)(25)

Dr Yang and Teng from China

In agreement is Dr Yang and Teng from China who published their study in 2007 European Journal of Endocrinology.(19) They studied three separate areas of China with low, moderate and excessively high Iodine intake and found the following:

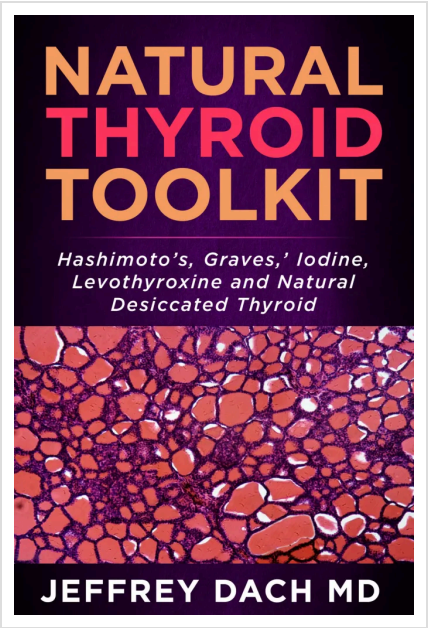
> *"long-term excessive iodine intake as an environmental factor does not seem to be involved in the occurrence of autoimmune hyperthyroidism."* (19)

Also in agreement is Dr. Zimmerman who found no increase in significant autoimmunity in 323 Moroccan children one year after an iodized salt program was introduced (20).

Conclusion: The take home message here was nicely said by Dr Dayan in his 1996 article in the NEJM.(21) He says:

> *"iodine reduces thyroid secretion in both subjects with thyroid autoantibodies and those without thyroid autoantibodies, suggesting that* ***it acts by inhibiting the biosynthesis and release of thyroid hormone rather than by augmenting thyroid autoimmunity.*** *"quote from Dayan(21)*

This suppressive effect of iodine can be used by the astute physician to treat hyperthyroidism in the auto-immune thyroid patient.



Natural Thyroid Toolkit

If you liked this article, you might like my new book, **Natural Thyroid Toolkit** available on Amazon. If you purchase a book, remember to leave a favorable review. That would be much appreciated. See the book cover, left image.

Update 9/19/21: BMJ Rapid Response:

Iodine deficiency, not excess, is the cause of autoimmune thyroid disease. The link between iodine intake and thyroid autoimmunity is more complex than Neeru Gupta suggests (Response, 08 April 2016), but increasing evidence implicates iodine deficiency, not excess, as the cause of autoimmune thyroid disease…. 12 April 2016 Peter J Lewis General Practitioner with Special Interest in Integrative Medicine 15 South Steyne, Manly, NSW 2095, Australia

Articles with related interest:

Iodine as a Treatment for Graves Hyperthyroidism

Iodine Selenium and Hashimotos

Selenium for Hashimotos PArt One

Hashimotos, Thyroid Disease and Molecular Mimicry

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Links and References

1) http://www.ncbi.nlm.nih.gov/pubmed/12728462 (free full text)
Yoon, Soo-Jee, et al. “The Effect of Iodine Restriction on Thyroid Function in Patients with Hypothyroidism Due to Hashimoto’s Thyroiditis.” Yonsei Med J 44.0 (2003): 2.

Yonsei Med J. 2003 Apr 30;44(2):227-35.
The effect of iodine restriction on thyroid function in patients with hypothyroidism due to Hashimoto’s thyroiditis.
Yoon SJ, Choi SR, Kim DM, Kim JU, Kim KW, Ahn CW, Cha BS, Lim SK, Kim KR, Lee HC, Huh KB.

Lifelong thyroid hormone replacement is indicated in patients with hypothyroidism as a result of Hashimoto’s thyroiditis. However, previous reports have shown that excess iodine induces hypothyroidism in Hashimoto’s thyroiditis. This study investigated the effects of iodine restriction on the thyroid function and the predictable factors for recovery in patients with hypothyroidism due to Hashimoto’s thyroiditis. The subject group consisted of 45 patients who had initially been diagnosed with hypothyroidism due to Hashimoto’s thyroiditis. The subjects were divided randomly into two groups. One group was an iodine intake restriction group (group 1) (iodine intake: less than 100 micro g/day) and the other group was an iodine intake non-restriction group (group 2). The thyroid-related hormones and the urinary excretion of iodine were measured at the baseline state and after 3 months. After 3 months, a recovery to the euthyroid state was found in 78.3 % of group 1 (18 out of 23 patients), which is higher than the 45.5% from group 2 (10 out of 22 patients). In group 1, mean serum fT4 level (0.80 +/- 0.27 ng/dL at the baseline, 0.98 +/- 0.21 ng/dL after 3 months) and the TSH level (37.95 +/- 81.76 micro IU/mL at the baseline, 25.66 +/- 70.79 micro IU/mL after 3 months) changed significantly during this period ($p < 0.05$). In group 2, the mean serum fT4 level decreased (0.98 +/- 0.17 ng/dL at baseline, 0.92 +/- 0.28 ng/dL after 3 months, $p < 0.05$). In the iodine restriction group, the urinary iodine excretion values were higher in the recovered patients than in non-recovered patients (3.51 +/- 1.62 mg/L vs. 1.21 +/- 0.39 mg/ L, $p=0.006$) and the initial serum TSH values were lower in the recovered patients than in the non-recovered patients (14.28 +/- 12.63 micro IU/mL vs. 123.14 +/- 156.51 micro IU/mL, $p=0.005$). In conclusion, 78.3% of patients with hypothyroidism due to Hashimoto’s thyroiditis regained an euthyroid state iodine restriction alone. Both a low initial serum TSH and a high initial urinary iodine concentration can be predictable factors for a recovery from hypothyroidism due to Hashimoto’s thyroiditis after restricting their iodine intake.

2) http://www.ncbi.nlm.nih.gov/pubmed/1951380
Am J Med. 1991 Oct;91(4):363-70. Natural history of thyroid abnormalities: prevalence, incidence, and regression of thyroid diseases in adolescents and young adults. Rallison ML, Dobyns BM, Meikle AW, Bishop M, Lyon JL, Stevens W.

This study reports the prevalence, incidence, and regression of thyroid abnormalities in a population observed from adolescence to adulthood.
PATIENTS AND METHODS: Examinations for thyroid abnormalities were performed in **4,819** school-age children, ages 11 to 18, in 1965 to 1968; two thirds of this original cohort (3,121) were re-examined 20 years later (1985 to 1986). Each subject with a thyroid abnormality detected by physical examination was studied by means of a series of re-examinations, and tests of thyroid function, imaging, and biopsy to determine the exact nature of the thyroid abnormality.
RESULTS:In the initial examinations (1965 to 1968), 185 thyroid abnormalities were found (3.7%). Diffuse hypertrophy with normal function (adolescent goiter) was the most common abnormality (19.3/1,000); 12.7/1,000 had chronic lymphocytic thyroiditis, and 4.6/1,000 had thyroid nodules, including two papillary carcinomas. Hyperthyroidism or hypothyroidism was found in 1.9/1,000. In the follow-up examinations in 1985 to 1986, 298 subjects had thyroid abnormalities (10.5%), of whom 81 (28.7/1,000) had simple goiters, 145 (51.3/1,000) had chronic thyroiditis, 45 (15.9/1,000) had hypothyroidism, 11 (3.9/1,000) had hyperthyroidism, and 66 (23.2/1,000) had nodules, which included 10 carcinomas. Of the 92 subjects with simple or adolescent goiter in 1965 to 1968, 60% were normal by 1985 to 1986, 20% were unchanged, and a few had developed thyroiditis (10%) or colloid goiters (3.0%). **Of 61 subjects with thyroiditis, 27% had become normal,** 33% remained unchanged, and 33% had become hypothyroid. Of the 22 subjects with thyroid nodules, two had complete disappearance of the nodules, and three had nodules considered to be variants of normal. The others exhibited a variety of nodular pathologic conditions. CONCLUSIONS: The natural history of thyroid disorders, including simple goiter, chronic thyroiditis, hyperthyroidism, hypothyroidism, and nodular diseases of the thyroid, indicates they are dynamic and changeable in form, function, appearance, and disappearance.

3) http://press.endocrine.org/doi/full/10.1210/jc.2002-020692
http://www.ncbi.nlm.nih.gov/pubmed/12466344
J Clin Endocrinol Metab. 2002 Dec;87(12):5499-502.
Effects of chronic iodine excess in a cohort of long-term American workers in West Africa. Pearce EN, Gerber AR, Gootnick DB, Khan LK, Li R, Pino S, Braverman LE. A cross-sectional survey of 102 Peace Corps volunteers in Niger, West Africa, in 1998 had previously demonstrated a high rate of thyroid dysfunction and goiter attributable to excess iodine from their water filters. The Peace Corps volunteers were followed-up a mean of 30 wk after they ceased using iodine-based water filtration systems. Goiter was present in 44% of subjects during excess iodine ingestion and in 30% after removal of excess iodine. Mean serum iodine decreased from **293 micro g/liter** during excess iodine ingestion to **84 micro g/liter** after cessation of excess iodine. Mean total serum T(4) values increased from 100.4 to 113.3 nmol/liter (7.8 to 8.8 micro g/dl). Mean serum free T(4) increased from 32.2 to 34.7 pmol/liter (2.5 to 2.7 ng/dl). **Mean serum TSH decreased from 4.9 to 1.8 mU/liter**. **Mean serum thyroid peroxidase antibody levels decreased from 33,000 to 22,000 IU/liter (33 to 22 IU/ml).** We found that during prolonged excess iodine exposure there were marked increases in serum total iodine concentrations, and the prevalence of goiter, elevated serum TSH values, and elevated serum thyroid peroxidase antibody values increased. The prevalence of all abnormalities decreased after removal of excess iodine from the drinking water system.

4) http://www.ncbi.nlm.nih.gov/pubmed/11086666
Aviat Space Environ Med. 2000 Nov;71(11):1120-5.
Thyroid function changes related to use of iodinated water in the U.S. Space Program. McMonigal KA, Braverman LE, Dunn JT, Stanbury JB, Wear ML, Hamm PB, Sauer RL, Billica RD, Pool SL.
The National Aeronautics and Space Administration (NASA) has used iodination as a method of microbial disinfection of potable water systems in U.S. spacecraft and long-duration habitability modules. A review of thyroid function tests of NASA astronauts who had consumed iodinated water during spaceflight was conducted.
METHODS:Thyroid function tests of all past and present astronauts were reviewed. Medical records of astronauts with a diagnosis of thyroid disease were reviewed. Iodine consumption by space crews from water and food was determined. Serum thyroid-stimulating hormone (TSH) and urinary iodine excretion from space crews were measured following modification of the Space Shuttle potable water system to remove most of the iodine.
RESULTS:**Mean TSH significantly increased in 134 astronauts who had consumed iodinated water during spaceflight.** Serum TSH, and urine iodine levels of Space Shuttle crewmembers who flew following modification of the potable water supply system to remove iodine did not show a statistically significant change. There was no evidence supporting association between clinical thyroid disease and the number of spaceflights, amount of iodine consumed, or duration of iodine exposure.
CONCLUSIONS:It is suggested that pharmacological doses of iodine consumed by astronauts transiently decrease thyroid function, as reflected by elevated serum TSH values. Although adverse effects of excess iodine consumption in susceptible individuals are well documented, exposure to high doses of iodine during spaceflight did not result in a statistically significant increase in long-term thyroid disease in the astronaut population.

5) http://www.ncbi.nlm.nih.gov/pubmed/21235106
Aviat Space Environ Med. 2011 Jan;82(1):49-51. Thyroid status of Space Shuttle crewmembers: effects of iodine removal. Smith SM, Zwart SR, McMonigal KA, Huntoon CL. Iodine is often used for water purification and has been used throughout the U.S. space program. Because of concern about potential effects on crewmembers' thyroid function, in 1997 a system was implemented on board the Space Shuttles to remove iodine from water before it was consumed. We report here thyroid hormone data from crews flying before and after this system was implemented.

Blood samples were collected and analyzed for thyroid hormone content during routine medical exams before and after Space Shuttle missions. Data are reported for 224 male and 49 female astronauts (about two-thirds of them before implementation of iodine removal).
RESULTS:Serum concentrations of total thyroxine (T4) and the free T4 index were elevated in men after flight and triiodothyronine (T3) was lower after flight, regardless of iodine removal status. T4 was higher, even before flight, in the group of men who flew after iodine removal was implemented. Conversely, T3 was lower in men who flew during that period. **Before iodine removal was implemented, thyroid stimulating hormone (TSH) was elevated in male and tended to be elevated in female astronauts, with average increases of 27% and 19% after flight, respectively.** After iodine removal was implemented, postflight TSH was not significantly different from preflight values.
DISCUSSION:These data provide evidence that crewmembers' increase in serum TSH on landing day after early Shuttle flights resulted from their consumption of iodinated water during spaceflight, because the same increase was not observed after implementation of the iodine removal system.

6) http://www.ncbi.nlm.nih.gov/pubmed/9761130
J Toxicol Environ Health A. 1998 Sep 25;55(2):93-106.
Comparison of the effects of iodine and iodide on thyroid function in humans. Robison LM, Sylvester PW,

Birkenfeld P, Lang JP, Bull RJ.
Concerns have been raised over the use of iodine for disinfecting drinking water on extended space flights. Most fears revolve around effects of iodide on thyroid function. iodine (I2) is the form used in drinking-water disinfection. Risk assessments have treated the various forms of iodine as if they were toxicologically equivalent. Recent experiments conducted in rats found that administration of iodine as I- (iodide) versus I2 had opposite effects on plasma thyroid hormone levels. I2-treated animals displayed elevated thyroxine (T4) and thyroxine/triiodothyronine (T/T3) ratios, whereas those treated with I- displayed no change or reduced plasma concentrations of T4 at concentrations in drinking water of 30 or 100 mg/L. The study herein was designed to assess whether similar effects would be seen in humans as were observed in rats. A 14-d repeated-dose study utilizing total doses of iodine in the two forms at either 0.3 or 1 mg/kg body weight was conducted with 33 male volunteers. Thyroid hormones evaluated included T4, T3, and thyroid-stimulating hormone (TSH). **TSH was significantly increased by the high dose of both I2 and I-, as compared to the control.** Decreases in T4 were observed with dose schedules with I- and I2, but none were statistically significant compared to each other, or compared to the control. This human experiment failed to confirm the differential effect of I2 on maintenance of serum T4 concentrations relative to the effect of I- that was observed in prior experiments in rats. However, **based on the elevations in TSH,** there should be some concern over the potential impacts of chronic consumption of iodine in drinking water.

7)http://www.ncbi.nlm.nih.gov/pubmed/16807415
N Engl J Med. 2006 Jun 29;354(26):2783-93. Effect of iodine intake on thyroid diseases in China. Teng W, Shan Z, Teng X, Guan H, Li Y, Teng D, Jin Y, Yu X, Fan C, Chong W, Yang F, Dai H, Yu Y, Li J, Chen Y, Zhao D, Shi X, Hu F, Mao J, Gu X, Yang R, Tong Y, Wang W, Gao T, Li C.
Iodine is an essential component of thyroid hormones; either low or high intake may lead to thyroid disease. We observed an increase in the prevalence of overt hypothyroidism, subclinical hypothyroidism, and **autoimmune thyroiditis with increasing iodine intake in China** in cohorts from three regions with different levels of iodine intake: mildly deficient (median urinary iodine excretion, 84 microg per liter), more than adequate (median, 243 microg per liter), and **excessive (median, 651 microg per liter**). Participants enrolled in a baseline study in 1999, and during the five-year follow-up through 2004, we examined the effect of regional differences in iodine intake on the incidence of thyroid disease.
METHODS:Of the 3761 unselected subjects who were enrolled at baseline, 3018 (80.2 percent) participated in this follow-up study. Levels of thyroid hormones and thyroid autoantibodies in serum, and iodine in urine, were measured and B-mode ultrasonography of the thyroid was performed at baseline and follow-up.
RESULTS:Among subjects with mildly deficient iodine intake, those with more than adequate intake, and those with excessive intake, the cumulative incidence of overt hypothyroidism was 0.2 percent, 0.5 percent, and 0.3 percent, respectively; that of subclinical hypothyroidism, 0.2 percent, 2.6 percent, and 2.9 percent, respectively; and that of **autoimmune thyroiditis, 0.2 percent, 1.0 percent, and 1.3 percent, respectively.** Among subjects with euthyroidism and antithyroid antibodies at baseline, the five-year incidence of elevated serum thyrotropin levels was greater among those with more than adequate or excessive iodine intake than among those with mildly deficient iodine intake. **A baseline serum thyrotropin level of 1.0 to 1.9 mIU per liter was associated with the lowest subsequent incidence of abnormal thyroid function.**
CONCLUSIONS:More than adequate or excessive iodine intake may lead to hypothyroidism and autoimmune thyroiditis.

8) http://www.ingentaconnect.com/content/aarm/jrm/2013/00000002/00000001/art00006
Flechas, Jorge. “Autoimmune Thyroiditis and Iodine Therapy.” Journal of Restorative Medicine 2.1 (2013): 54-59.

9) http://www.optimox.com/pics/Iodine/IOD-22/IOD_22.htm#2
Facts about Iodine and Autoimmune Thyroiditis by Guy E. Abraham, MD

10) http://www.ncbi.nlm.nih.gov/pubmed/12800544
Endocrinol Metab Clin North Am. 2003 Jun;32(2):519-34.
Perioperative management of the thyrotoxic patient. Langley RW, Burch HB. Preoperative thyrotoxicosis is a potentially life-threatening condition that requires medical intervention before surgery. Most patients are undergoing thyroidectomy for persistent thyrotoxicosis, usually Graves' disease, either having contraindications to or failing medical therapy. Fewer patients are undergoing nonthyroidal surgery that is likely urgent or emergent. The choice of treatment depends on the time available for preoperative preparation, the severity of the thyrotoxicosis, and the impact of any current or previous therapies. Generally treatment is directed at a combination of targets in the thyroid hormone synthetic, secretory, and peripheral pathway with concurrent treatment to correct any decompensation of normal homeostatic mechanisms. Thionamides are the preferred initial treatment unless contraindicated, but do require several weeks to render a patient euthyroid. **Beta-Blockers** should always be used unless absolutely contraindicated because they improve thyrotoxic symptoms especially of the cardiovascular system. Other agents including **iodine** and steroids can be used if rapid preparation is required or more severe thyrotoxicosis is present. The goal of therapy is to render the patient as close as possible to clinical and biochemical euthyroidism before surgery. Overall, the morbidity and mortality of adequately prepared patients is low.

11) http://www.ncbi.nlm.nih.gov/pubmed/3317958
Surgery. 1987 Dec;102(6):1055-61.
The effect of preoperative Lugol's iodine on thyroid blood flow in patients with Graves' hyperthyroidism. Chang DC, Wheeler MH, Woodcock JP, Curley I, Lazarus JR, Fung H, John R, Hall R, McGregor AM.
A study was conducted to investigate the effect of **Lugol's iodine** on the superior thyroid artery (STA) blood flow with use of a Duplex ultrasound scanner for **12 patients with Graves' disease**. All patients were treated with antithyroid drugs until they were euthyroid and then, with randomization, the patients received either Lugol's iodine, 0.3 ml thrice daily, or placebo for 9 days in a double-blind fashion. Antithyroid drugs were continued throughout the study. Reduction in the diameter, time-averaged velocity (TAV), and volume flow (VF) of the STAs was demonstrated in all patients in the treatment group, whereas there were no consistent trends in the placebo group. The changes in TAV and VF were significantly different between the placebo and treatment groups (p less than 0.01 for TAV and p less than 0.005 for VF). These changes were more marked in patients with high initial VF and minimal in patients with low initial VF. **On the basis of these results, we recommend that patients with high thyroid blood flow before thyroidectomy should receive Lugol's iodine preoperatively.**

12) http://www.ncbi.nlm.nih.gov/pubmed/9752450
Ann Chir. 1998;52(3):229-33.
[Effect of preoperative administration of Lugol's solution on thyroid blood flow in hyperthyroidism]. [Article in French]
Rodier JF, Janser JC, Petit H, Schneegans O, Ott G, Kaissling A, Grob JC, Velten M.
A study of **50 patients with hyperthyroidism** was conducted to evaluate the effect of preoperative administration of Lugol's iodine solution on thyroid blood flow. Highly significant reductions in diameter, time-averaged velocity, and volume flow of the superior thyroid artery were demonstrated after administration of Lugol's solution. The Duplex ultrasound scanning used in this study is a noninvasive, inexpensive, accurate, and reproducible technique suitable for analysis of thyroid blood flow in hyperthyroidism. On the basis of current ultrasonographic results and

low postoperative morbidity in patients, Lugol's solution is well tolerated and may be recommended for use before thyroidectomy, **especially for diffuse toxic goiters and Graves disease**.

13) http://www.ncbi.nlm.nih.gov/pubmed/2457351
Ann R Coll Surg Engl. 1988 May;70(3):123-7.
Effect of preoperative iodine in patients with Graves' disease controlled with antithyroid drugs and thyroxine.
Kaur S, Parr JH, Ramsay ID, Hennebry TM, Jarvis KJ, Lester E.
Thirty-four patients with Graves' disease, first rendered euthyroid with antithyroid drugs (ATD) then given supplementary thyroxine (T4), were randomly allocated to three treatment groups. In Group I ATD and T4 were stopped 10 days before partial thyroidectomy and the patients were treated with **Lugol's iodine alone**. In Group II the patients were treated up to the time of operation with ATD and T4 alone. In Group III ATD and T4 were continued until the day of operation, but the patients also received 10 days' treatment with Lugol's iodine. Analysis of the results showed that pre-operative iodine therapy in patients with Graves' disease, already rendered euthyroid with ATD and T4, made no difference to the vascularity of the gland, operative blood loss or thyroid follicular size. Over a third of patients in Group I, treated with Lugol's iodine alone for 10 days pre-operatively, had subnormal levels of thyroid hormones at the time of operation and this was also the Group in which the complications of partial thyroidectomy tended to occur. **It is concluded that the addition of iodine preoperatively is unnecessary in the patient who is already euthyroid on ATD and T4**.

14) http://www.ncbi.nlm.nih.gov/pubmed/12213665
Eur J Endocrinol. 2002 Sep;147(3):293-8.
Effect of iodine or iopanoic acid on thyroid Ca2+/NADPH-dependent H2O2-generating activity and thyroperoxidase in toxic diffuse goiters. Cardoso LC, Martins DC, Campos DV, Santos LM, Corrêa da Costa VM, Rosenthal D, Vaisman M, Violante AH, Carvalho DP.
The aim of the present study was to compare the effects of iopanoic acid (IOP) or a saturated solution of **potassium iodide** (SSKI) administration to patients with toxic diffuse goiters (TDG).
DESIGN:Patients with TDG are treated with thionamides and high doses of iodine preoperatively. In this study, two types of preoperative drug regimens were used: propylthiouracil or methimazole plus SSKI for 10-15 days (n=8) or IOP for 7 days (n=6).
METHODS:Serum thyroid hormones (total and free thyroxine (T(4)), total tri-iodothyronine (T(3)) and reverse T(3) (rT(3)), were evaluated after 7 days of either SSKI or IOP treatment, and after 10-15 days of SSKI administration. During thyroidectomy, samples of thyroid gland were obtained to evaluate thyroperoxidase and thyroid H(2)O(2)-generating activities.
RESULTS:**Serum total T(3) was significantly decreased after 7 days of either treatment**, and serum rT(3) was significantly increased in IOP-treated patients. Serum total and free T(4) were unaffected by 7 days of IOP treatment, but decreased after 7 days of SSKI treatment, although significantly diminished levels were only reached after a further 3-8 days of SSKI administration. During both drug regimens, serum TSH remained low (SSKI: 0.159+/-0.122; IOP: 0.400+/-0.109 microU/ml). **Thyroperoxidase activity was significantly lower in thyroid samples from patients treated with SSKI for 10-15 days than in the thyroid glands** from IOP-treated patients. However, thyroid H(2)O(2) generation was inhibited in samples from patients treated with either IOP or SSKI.
CONCLUSIONS:**We show herein that IOP treatment can be effective in the management of hyperthyroidism and that this drug inhibits thyroid NADPH oxidase activity, just as previously described for SSKI**, probably due to its **iodine** content.

15) johansson_janet_110606_iodine_selenium_Hashimoto_dogs
Swedish University of Agricultural Sciences
Faculty of Veterinary Medicine and Animal Science
Review of the literature and an attempt to evaluate intake levels of iodine and selenium in dogs with and without lymphocytic thyroiditis
Janet Johansson Master's Thesis, 30 HEC 2011

16) http://www.ncbi.nlm.nih.gov/pubmed/9703374 (free full text)
Eur J Endocrinol. 1998 Jul;139(1):23-8.
Effect of small doses of iodine on thyroid function in patients with Hashimoto's thyroiditis residing in an area of mild iodine deficiency.
Reinhardt W, Luster M, Rudorff KH, Heckmann C, Petrasch S, Lederbogen S, Haase R, Saller B, Reiners C, Reinwein D, Mann K.
Several studies have suggested that iodine may influence thyroid hormone status, and perhaps antibody production, in patients with autoimmune thyroid disease. To date, studies have been carried out using large amounts of iodine. Therefore, we evaluated the effect of **small doses of iodine on thyroid function and thyroid antibody levels in euthyroid patients with Hashimoto's thyroiditis** who were living in an area of **mild dietary iodine deficiency.**
METHODS:Forty patients who tested positive for anti-thyroid (TPO) antibodies or with a moderate to severe hypoechogenic pattern on ultrasound received **250 microg potassium iodide daily for 4 months** (range 2-13 months).

An additional 43 patients positive for TPO antibodies or with hypoechogenicity on ultrasound served as a control group. All patients were TBII negative.

RESULTS:Seven patients in the iodine-treated group developed **subclinical hypothyroidism** and one patient became hypothyroid. **Three of the seven who were subclinically hypothyroid became euthyroid again when iodine treatment was stopped. One patient developed hyperthyroidism with a concomitant increase in TBII titre to 17 U/l, but after iodine withdrawal this patient became euthyroid again**. Only one patient in the control group developed subclinical hypothyroidism during the same time period. All nine patients who developed thyroid dysfunction had reduced echogenicity on ultrasound. Four of the eight patients who developed subclinical hypothyroidism had TSH concentrations greater than 3 mU/l. **In 32 patients in the iodine-treated group and 42 in the control group, no significant changes in thyroid function, antibody titres or thyroid volume were observed.**
CONCLUSIONS:Small amounts of supplementary iodine (250 microg) cause slight but significant changes in thyroid hormone function in predisposed individuals.

17) http://eje-online.org/content/158/2/209.long
Eur J Endocrinol. 2008 Feb;158(2):209-15. Serum TSH and serum thyroid peroxidase antibody fluctuate in parallel and high urinary iodine excretion predicts subsequent thyroid failure in a 1-year study of patients with untreated subclinical hypothyroidism. Karmisholt J, Laurberg P.
To explore the possibility of predicting decline or improvement in thyroid function over 1 year, and to investigate the correlations of serum TSH (s-TSH) with hypothyroidism-related symptoms and signs, serum thyroid peroxidase antibody (s-TPO-Ab) and urinary iodine excretion in individual patients with untreated subclinical hypothyroidism (SH).
DESIGN:Monthly repeated measurement study without intervention.

METHODS:**Twenty-on**e patients without former thyroid disease who had been identified with s-TSH between **5 and 12** mU/l and normal serum thyroxine (s-T4) at two occasions were enrolled. **Subsequently, 13 monthly measurements of s-TSH, hypothyroidism-related symptoms and signs, serum free T4, s-TPO-Ab and urinary iodine excretion were performed.**
RESULTS:Over the study year, s-TSH increased significantly in 5 patients, 16 had unchanged s-TSH, whereas none improved. From clinical and biochemical inclusion data, it was not possible to predict who would later increase in s-TSH. In individual patients, a highly significant correlation between s-TSH and s-TPO-Ab was found ($r=0.37$, $P<0.0001$) and also between s-TSH and urinary iodine excretion ($r=0.14$, $P=0.034$). No correlation between s-TSH and clinical symptoms and signs was observed. Time shift showed best correlation between s-TSH and s-TPO-Ab measured at the same time point, whereas urinary iodine excretion correlated best to s-TSH and s-TPO-Ab obtained 1 month later.
CONCLUSION:**At the time of inclusion, it was not possible to identify the 24% of SH patients who would show deterioration in thyroid function over the following year.** Impairment in thyroid function varied in parallel with thyroid autoimmunity, whereas high urinary iodine excretion predicted **high s-TSH** and **s-TPO-Ab 1 month later**.

18) http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3195970/
Croat Med J. 2011 Oct 15;52(5):615-21.
Ten-year follow-up of thyroid epidemiology in Slovenia after increase in salt iodization. Zaletel K, Gaberscek S, Pirnat E.
To assess iodine supply and follow thyroid epidemiology for ten years after an iodine increase from 10 to 25 mg of potassium iodide per kilogram of salt in 1999. METHODS:In 2002 and 2003, we determined the thyroid size by palpation and ultrasound and measured urinary iodine concentration (UIC) in 676 schoolchildren from 34 schools throughout Slovenia. From 1999 to 2009, we followed the incidence of diffuse and nodular goiter, thyroid autonomy, Graves' disease, and Hashimoto's thyroiditis among adults in the stable catchment area of the University Medical Centre Ljubljana with 1000000 inhabitants. RESULTS.:In children, only 1% had a goiter grade 2 (visible and palpable thyroid gland), median thyroid volume was 5.8 mL, and median **UIC was 148** μg/L. In adults, the incidence of diffuse goiter and thyroid autonomy decreased significantly (2009 vs 1999, rate ratio [RR], 0.16; 95% confidence interval [CI], 0.12-0.21 and RR, 0.73; 95% CI, 0.62-0.86, respectively), with a lower incidence in younger participants in 2009 ($P<0.001$). **The incidence of multinodular goiter and solitary nodule increased** (2009 vs 1999, RR, 1.55; 95% CI, 1.35-1.79 and RR, 1.72; 95% CI, 1.49-1.99, respectively). No long-term changes were observed for Graves' disease (2009 vs 1999, RR, 0.95; 95% CI, 0.81-1.13), **while the incidence of Hashimoto's thyroiditis increased strongly** (2009 vs 1999, RR, 1.86; 95% CI, 1.64-2.12).
CONCLUSIONS:The change from mildly deficient to sufficient iodine supply was associated with a marked change in the incidence of thyroid epidemiology – a significant decline in the incidence of diffuse goiter and thyroid autonomy and **a marked increase in the incidence of Hashimoto's thyroiditis.**

19) http://eje-online.org/content/156/4/403.long
http://www.ncbi.nlm.nih.gov/pubmed/17389453
Eur J Endocrinol. 2007 Apr;156(4):403-8.
Chronic iodine excess does not increase the incidence of hyperthyroidism: a prospective community-based epidemiological survey in China. Yang F, Shan Z, Teng X, Li Y, Guan H, Chong W, Teng D, Yu X, Fan C, Dai H, Yu Y, Yang R, Li J, Chen Y, Zhao D, Mao J, Teng W.
An increasing incidence of hyperthyroidism has been observed when iodine supplementation has been introduced to an iodine-deficient population. Moreover, the influence of chronic more than adequate or excessive iodine intake on the epidemiological features of hyperthyroidism has not been widely and thoroughly described. To investigate

the influences of different iodine intake levels on the incidence of hyperthyroidism, we conducted a prospective community-based survey in three communities with mild-deficient, more than adequate (previously mild deficient iodine intake), and excessive iodine intake.
SUBJECTS AND METHODS:In three rural Chinese communities, a total of 3761 unselected inhabitants aged above 13 years participated in the original investigation and 3018 of them received identical examinations after 5 years. Thyroid function, levels of thyroid peroxidase antibody (TPOAb), thyroglobulin antibody and urinary iodine excretion were measured and thyroid ultrasound examination was also performed.
RESULTS:In three communities, median urinary iodine excretion was 88, 214, and 634 microg/l (P<0.05) respectively. The cumulative incidence of hyperthyroidism was 1.4, 0.9, and 0.8% (P>0.05) respectively. Autoimmune hyperthyroidism was predominant in thyroid hyperfunction in all the three cohorts. Either positive TPOAb (>50 U/ml) or goiter in original healthy participants was associated with the occurrence of unsuspected hyperthyroidism in 5 years (logistic regression, OR=4.2 (95% CI 1.7-8.8) for positive TPOAb, OR=3.1 (95% CI 1.4-6.8) for goiter).
CONCLUSION:Iodine supplementation may not induce an increase in hyperthyroidism in a previously mildly iodine-deficient population. Chronic iodine excess does not apparently increase the risk of autoimmune hyperthyroidism, suggesting that excessive iodine intake may not be an environmental factor involved in the occurrence of autoimmune hyperthyroidism.

“long-term excessive iodine intake as an environmental factor does not seem to be involved in the occurrence of autoimmune hyperthyroidism. ”

20) http://www.ncbi.nlm.nih.gov/pubmed/12699595
Thyroid. 2003 Feb;13(2):199-203.
Introduction of iodized salt to severely iodine-deficient children does not provoke thyroid autoimmunity: a one-year prospective trial in northern Morocco.Zimmermann MB, Moretti D, Chaouki N, Torresani T.
To determine if introduction of iodized salt induces thyroid autoimmunity in goitrous children, we conducted a prospective trial in iodine-deficient Moroccan schoolchildren (n = 323). Local salt was iodized at 25 microg iodine per gram of salt and distributed to households. Before introduction of iodized salt and at 10, 20, 40, and 52 weeks, we measured antithyroid peroxidase antibodies (TPO-Ab), antithyroglobulin antibodies (Tg-Ab), urinary iodine (UI), and thyroid hormones, and examined the thyroid using ultrasound. At baseline, median UI was 17 microg/L and the prevalence of goiter and hypothyroidism was 72% and 18%, respectively. Provision of iodized salt maintained median UI at 150-200 microg/L for the year ($p < 0.0001$). There was a significant increase in mean total thyroxine (T(4)) and a significant reduction in the prevalence of hypothyroidism ($p < 0.001$). There was a transient increase in the prevalence of detectable antibodies after introduction of iodized salt ($p < 0.0001$) with levels returning to baseline at 1 year. Only congruent with 1% of children had elevated TPO-Ab and none had elevated Tg-Ab over the course of the study, and no child with elevated TPO-Ab had abnormal thyrotropin (TSH) or T(4) concentrations. None developed clinical or ultrasonographic evidence of thyroid autoimmune disease and/or iodine-induced hypothyroidism or hyperthyroidism. Rapid introduction of iodized salt does not provoke significant thyroid autoimmunity in severely iodine-deficient children followed for 1 year.

21) Autoimmune_Thyroiditis_Dayan_NEJM_1996 (Full pdf)
http://www.ncbi.nlm.nih.gov/pubmed/8649497
N Engl J Med. 1996 Jul 11;335(2):99-107.
Chronic autoimmune thyroiditis.
Dayan CM, Daniels GH.
However, iodine reduces thyroid secretion in both subjects with thyroid autoantibodies and those without thyroid

autoantibodies, suggesting that it acts by inhibiting the biosynthesis and release of thyroid hormone rather than by augmenting thyroid autoimmunity. 47,48

22) (full pdf)
2003_Kasagi_Effect_of_Iodine_Restriction_on_Thyroid_Function_in_Thyroid

http://www.ncbi.nlm.nih.gov/pubmed/12930600
Thyroid. 2003 Jun;13(6):561-7.
Effect of iodine restriction on thyroid function in patients with primary hypothyroidism. Kasagi K, Iwata M, Misaki T, Konishi J.
Dietary iodine intake in Japan varies from as little as 0.1 mg/day to as much as 20 mg/day. The present study was undertaken to assess the frequency of iodine-induced reversible hypothyroidism in patients diagnosed as having primary hypothyroidism, and to clarify the clinical backgrounds responsible for the spontaneous recovery of thyroid functions. Thirty-three consecutive hypothyroid patients (25 women and eight men) with a median age of 52 years (range, 21-77 years) without a history of destructive thyroiditis within 1 year were asked to refrain from taking any iodine-containing drugs and foods such as seaweed products for 1-2 months. The median serum thyrotropin (TSH) level, which was initially 21.9 mU/L (range, 5.4-285 mU/L), was reduced to 5.3 mU/L (range, 0.9-52.3 mU/L) after iodine restriction. Twenty-one patients (63.6%) showed a decrease in serum TSH by >50% and to <10 mU/L. Eleven patients (33.3%) became euthyroid with TSH levels within the normal range (0.3-3.9 mU/L). The ratios of TSH after iodine restriction to TSH before iodine restriction (aTSH/bTSH) did not correlate significantly with titers of anti-thyroid peroxidase antibody and anti-thyroglobulin antibody or echogenicity on ultrasonography, but correlated inversely with (99m)Tc uptake ($r = 0.600$, $p < 0.001$). Serum non-hormonal iodine levels, although not correlated significantly with aTSH/bTSH values, were significantly higher in the 21 patients with reversible hypothyroidism than in the remaining 12 patients. TSH binding inhibitor immunoglobulin was negative in all except one weakly positive case. In conclusion, (1) primary hypothyroidism was recovered following iodine restriction in more than half of the patients, and (2) the reversibility of hypothyroidism was not significantly associated with Hashimoto's thyroiditis but with increased (99m)Tc uptake and elevated non-hormonal iodine levels.

Full pdf of article:

23) 2012_Leung_Sufficient_iodine_intake_during_pregnancy_Just_do_it_Thyroid

http://www.ncbi.nlm.nih.gov/pubmed/23134529
Thyroid. January 2013, 23(1): 7-8. Sufficient Iodine Intake During Pregnancy: Just Do It no access. Angela M. Leung, Elizabeth N. Pearce, Lewis E. Braverman

According to data from the National Health and Nutrition Examination Survey (NHANES), the median urinary iodine concentration in U.S. adults decreased by over 50% from the early 1970s to the late 1990s (10). Of particular concern in the NHANES data is that the prevalence of urinary iodine values < 50 lg/L among women of childbearing age increased by almost fourfold, from 4% to 15%, during this period. The most recent NHANES survey (2005–2008) demonstrated that 35.3% of pregnant women had urinary

iodine levels below 100 lg/L (11), which suggests mild iodine deficiency (12). Reductions in U.S. dietary iodine have been variously ascribed to a possible reduction in the iodine content of dairy products, the removal of iodate dough conditioners in commercially produced bread, new recommendations for reduced salt intake for blood pressure control, the recent increased use of Kosher and sea salts (which contain no iodine), and the increasing use of noniodized salt in manufactured or ‘‘pre-made’’ convenience foods (13).

NHANES Survey showing 50% decline in Iodine levels
24) http://www.ncbi.nlm.nih.gov/pubmed/9768638
Hollowell JG, Staehling NW, Hannon WH, Flanders DW, Gunter EW, Maberly GF, Braverman LE, Pino S, Miller DT, Garbe PL, DeLozier DM, Jackson RJ 1998 Iodine nutrition in the United States. Trends and public health implications: iodine excretion data from National Health and Nutrition Examination Surveys I and III (1971–1974 and 1988–1994). J Clin Endocrinol Metab 83:3401–3408.

25) U-Tube Video Presentation: David Brownstein MD – Does Iodine Cause, Worsen, Treat or Improve Autoimmune Thyroid

Images courtesy of :

http://www.thyroidmanager.org/chapter/hashimotos-thyroiditis/
Hashimoto’s Thyroiditis Last Updated: December 20, 2013 Authors Takashi Akamizu, M.D., Ph.D. Professor and Chairman, The First Department of Medicine, Wakayama Medical University, 811-1 Kimi-idera, Wakayama 641-8509, Japan, Nobuyuki Amino, M.D. Kuma Hospital, Center for Excellence in Thyroid Care, 8-2-35 Shimoyamate-dori, Chuo-ku, Kobe 650-0011, Japan, Leslie J. DeGroot, M.D. Research Professor, CELS, University of Rhode Island, 80 Washington St, Providence, RI 02903

1) Electron microscopy image of thyroid tissue from a patient with Hashimoto’s thyroiditis, showing electron dense deposits of IgG and TG along the basement membrane of follicular cells.

2) Pathology of Hashimoto’s thyroiditis. In this typical view of severe Hashimoto’s thyroiditis, the normal thyroid follicles are small and greatly reduced in number, and with the hematoxylin and eosin stain are seen to be eosinophilic. There is marked fibrosis. The dominant feature is a profuse mononuclear lymphocytic infiltrate and lymphoid germinal center formation.

3) Normal Thyroid Gland Histology Image

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The reader is advised to discuss the comments on these pages with his/her personal physicians and to only act upon the advice of his/her personal physician. Also note that concerning an answer which appears as an electronically posted question, I am NOT creating a physician — patient relationship. Although identities will remain confidential as much as possible, as I can not control the media, I can not take responsibility for any breaches of confidentiality that may occur.

Link to this article:http://wp.me/p3gFbV-1eq

Summary

This entry was posted in **autoimmune disease**, **Hashimotos**, **Hypothyroidism**, **Iodine**, **jeffrey dach**, **Levothyroxine**, **selenium**, **Thyroid**, **TSH** and tagged **Antibody**, **autoimmune**, **hashimotos**, **iodine**, **Reinhardt**, **Thyroid Peroxidase**, **TPO**, **Yoon** by **Jeffrey Dach MD**. Bookmark the **permalink [https://jeffreydachmd.com/2014/02/iodine-hashimotos-autoimmune-thyroid-disease/]** .

17 THOUGHTS ON "IODINE AND HASHIMOTO'S AUTOIMMUNE THYROID DISEASE"

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SEED
on **May 3, 2016 at 12:18 AM** said:

Thank you for the studies and analysis. I do appreciate those who warn of potential hazards involving iodine supplementation, but this analysis is very persuasive.

Anne
on **April 29, 2017 at 3:17 PM** said:

Thank you so much for providing this valuable information. I learned a lot. Please forgive me, as I am not able to understand the full scope of all these studies and reports. My question to you is; Should people with Hashimotos take iodine or not? I have been advised in both diections and remain confused about this. Please help me understand, I really want to know. My last antibody count was 59 and was told by one doctor to avoid iodine, and told by another doctor to take it. Please help me. Thank you!

Qur'an 18:86
on **May 10, 2017 at 4:54 PM** said:

The general point is that iodine in doses up to about 50mg (50,000mcg) won't cause Hashi provided you already have sorted out sufficient Selenium status.

Hema
on **July 4, 2017 at 9:55 PM** said:

Thank you so much for posting this.i was very confused about whether or not to take iodine. After painting about 10 drops for 2 days. I stared feeling worse. This post is very informative, I think people with hashimotos should not take iodine. If you do as advised by iodine doctors and advocates it has to be higher doses and be prepared for a transient increase in TSH and all the hypothyroid symptoms. Any comments on this ?

Pingback: 橋本氏甲狀腺炎會增加癌症風險嗎？ – 華悅身心靈健康中心

Ericka
on **March 19, 2018 at 8:47 AM** said:

The Yoon study was restricted to less than 100 mcg per day.
https://www.ncbi.nlm.nih.gov/pubmed/12728462

jeffrey_dach_md
on **April 3, 2018 at 6:26 PM** said:

Hi Ericka,

Yes this is stated, however, actual iodine intake was higher as reported in the bod of the text.